block with slow pulse, however, the same writers failed to note any increase of ventricular rate after the use of moderately large doses of digitalis. It is possible that the appearance of extrasystoles and the temporary disappearance of the *a* waves from the jugular pulse (due to a toxic weakening of the auricular contractions?) may have been due to the drug.

Auricular Fibrillation.—It is well known that in the latest stages of cardiovascular degeneration, especially in mitral stenosis, the pulse often becomes exceedingly irregular, and in the jugular the wave of auricular contraction disappears. This has long been regarded, particularly by Mackenzie, as depending upon the origin of the rhythm at the node of Tawara (hence the term nodal rhythm). LEWIS (*Brit. Med. Jour.*, 1909, ii) asserts that facts are at his disposal permitting the conclusion that the rhythm arising in the neighborhood of node gives rise to a different clinical picture. This conclusion is based upon the study of an instance of paroxysmal tachycardia in which auricle and ventricle contract together. Secondly, the pulsus irregularis perpetuus is dependent upon fibrillation of the auricle. This conclusion is based upon the fact that the rhythm is exactly similar to that which may be produced experimentally by inducing fibrillation of the auricle, and is a unique condition. Lewis points to the fact that electrocardiograms taken from patients exhibiting this irregularity, show a number of irregular waves apart from the ventricular curve, and more clearly defined in diastole. Such waves are found in no other disorder of the heart action. They disappear when irregularity vanishes, are not evident upon the cardiogram, and are identical with the curves yielded by fibrillation of the auricle. Furthermore, synchronous tracings show that the waves in the experimental cardiogram correspond to the fibrillary movements of the auricle. [In connection with this interesting communication it may be remembered that Cushny and Edwards in the AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1907, cxxxiii, 66, arrive at the conclusion that an instance of paroxysmal irregularity was probably due to this cause.—W. S. T.]

The Etiology of Beri-beri.—The studies of FRASER and STANTON at the Institute for Medical Research, Federated Malay States (*Trans. Soc. Tropical Med. and Hygiene*,1910, iii, 257), are based on the chemical analyses of various types of rice, and on the production of polyneuritis gallinarum, a disease analogous to beri-beri, by feeding experiments in fowls. It was first found that Siam rice, which is most often associated with epidemics of beri-beri, contains a lower percentage of fat, than either Rangoon rice or parboiled rice. Microscopic sections showed that in Siam rice the pericarp, the outer layer, containing most of the aleurone and oily material, had been removed by the process of polishing. The relation of the milling of rice to the production of the disease in fowls was then studied. Fowls fed on the original padi ale remained healthy. Of twelve fowls fed on the finished, polished rice, six developed polyneuritis. Other fowls fed on the same finished rice, plus the polishings, all remained healthy. From these experiments Fraser and Stanton concluded that the polishing of white rice removes from the seed some substance essential to the maintenance of the normal nutrition of nerve tissues. It was further shown that staleness of rice, or the development in it of poisonous substances subsequent to its being milled are not important factors. Parboiled rice, in itself healthy, when extracted with alcohol, caused polyneuritis in fowls, but the addition of the alcoholic extract to a rice known to be injurious prevented this disease. Further chemical investigations showed that the power of a rice to produce polyneuritis gallinarum varied with its phosphorus content—the higher the phosphorus content, the less liable was it to be injurious. The highest percentage of phosphorus was found to be present in rice polishings. Moreover, the addition to an injurious rice of a quantity of polishings which contained enough phosphorus to bring the total phosphorus content up to that of parboiled rice sufficed to preserve nutritive equilibrium. The prevention of beri-beri thus depends on the substitution of ordinary white rice, by a rice in which the polishing process has been omitted, or carried out to a minimal extent, or by the addition to a white rice diet of articles rich in those substances which are not present in sufficient amount in white rice. One such article, which is cheap, and may be readily obtained, is the polishings from white rice.

The Physiology of the Immediate Reaction of Anaphylaxis.—On the injection of a dose of horse serum into the vein of a previously highly sensitive guinea-pig, there occurs a chain of symptoms—chiefly of respiratory nature, which result in the death of the animal in from three to five minutes. While the general type of the reaction has been reported and confirmed by various observers, it has remained for AUER and LEWIS (*Jour. Exper. Med.*, 1910, xii, 151) to study and explain the physiological basis. While convulsive and paralytic symptoms may dominate the picture if the animal is loose, they found that if it is held in a suitable holder these are less marked and the respiratory changes come into the foreground. They thus paid especial attention to the lungs, and found, as did Gay and Southard, that at autopsy the lung in acute anaphylaxis tends to remain in an inspiratory, distended condition with open thorax, with unobstructed trachea, and large bronchi, and without obvious pulmonary œdema. This immobilization of the lungs they consider to be the most characteristic sign of immediate anaphylaxis in the guinea-pig. Experiments were performed in which the respiratory movements of the chest and the volume changes of the pleural cavity were recorded, as well as others in which the animals were allowed to breathe from a bottle, and changes during inspiration and expiration registered. As a result it was evident that some stenosis is gradually produced in the pulmonary passages so that in the final stage practically no air enters or leaves the lung in spite of violent respiratory attempts. Death is due to asphyxia. The characteristic reaction of anaphylaxis was also obtained in pithed animals, showing that its production depends on a peripheral process in the lung, and not on the central nervous system. After reviewing the possible cause of the condition, the authors conclude that it is due to a tetanic contracttion of the muscles of the finer bronchioles, so that air is imprisoned in the areolar sacs. Atropine, which paralyzes the bronchial muscles, may, under certain conditions, be able to relax the anaphylactic lung so that it is again able to expand and contract. The blood pressure